913-127 Alteration of Typical Atrial Flutter Circuit After Apparent Bi-directional Conduction Block Through the Isthmus Between the Inferior Vena Cava and Tricuspid Annulus

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During typical atrial flutter (AFL) counterclockwise conduction proceeds through an atrial isthmus between the inferior vena cava and tricuspid annulus (IVC-TA), resulting in posterior atrial septal activation preceding anterior activation. Concealed entrainment of typical AFL is observed from multiple locations, including the IVC-TA isthmus and within the proximal coronary sinus ostium (CSO). Bi-directional conduction block during sinus rhythm through this isthmus after ablation has been an indicator of successful elimination of the AFL circuit. However, we report results from a group of pts with typical AFL who exhibited a new type of counterclockwise AFL after successful ablation of their typical AFL and despite production of apparent conduction block within their isthmus. Standard intracardiac electrophysiologic techniques were utilized. In 15 consecutive pts with typical AFL, bi-directional conduction block through the IVC-TA isthmus was documented after successful ablation of typical AFL. However, in 6/15 pts, a new counterclockwise AFL could be induced with anterior septal activation preceding posterior septal activation. In 2/6 pts this activation shift occurred during ablation without termination of the tachycardia suggesting the new AFL was merely an altered form of the original typical AFL. Unlike the original typical AFL, concealed entrainment during this new counterclockwise AFL could not be demonstrated from within the CSO; proximal CSO pacing resulted only in manifest entrainment or dissociation from the tachycardia. However, in these 2 pts concealed entrainment could be produced just posterior to the CSO and linear ablation from the CSO to IVC rim terminated this unique AFL. In conclusion, our results suggest that in some pts, while conduction block within the IVC-TA isthmus portion of typical AFL circuit does eliminate typical AFL, it may also isolate the CSO creating a new critical isthmus between the CSO and IVC allowing the development of a new counterclockwise tachycardia which may be ablated by targeting this IVC-CSO isthmus.

913-128 Incidence of Atrial Arrhythmias in patients with Proven Isthmus Block after Radiofrequency Ablation of Inferior Vena Cava-Tricuspid Ring for Common Atrial Flutter

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Introduction: Common type of atrial flutter (Afl) is now amenable to catheter radiofrequency ablation (RFA) with a high acute and late success rate. Creation of a bi-directional block at the inferior vena cava-tricuspid annulus isthmus (IVC-TAI) or at the tricuspid annulus-eustachian ridge isthmus (TA-ERI) prevents against recurrences. However, little is known about the incidence of other atrial arrhythmias following RFA.

Methods and Results: We analyzed the late outcome of 37 patients (pts) who underwent RFA of Afl with confirmed acute creation of a complete bi-directional IVC-TAI or TA-ERI block. Before RFA, 14 pts presented with documented episodes of atrial fibrillation (Afib) in addition to Afl which however remained the major symptomatic arrhythmia. Follow-up consisted of regular outpatient's consultations with ECGs and 24 hour Holter monitoring. In ten cases, control electrophysiologic study confirmed the late presence of isthmus block. Over a mean follow-up duration of 16.7 ± 5.2 months (range 9–33), only 12 pts (32%) complained of persistent palpitations which were found to be correlated to arrhythmia episodes in nine. Fifteen pts had documented episodes of Afib documentation alternating with episodes of a typical atrial flutter in 2. Seven of the 14 pts (50%) previously suffering from Afib were totally free of arrhythmia. Out of 23 pts without Afib before RFA (30%), 8 showed Afib episodes after the procedure. No recurrence of common Afl was documented.

Conclusions: A significant number of patients still complain of sustained palpitations despite confirmed bi-directional IVC-TAI or TA-ERI block. These are mostly due to Afib but not to Afl recurrence. On the other hand, RFA of IVC-TAI or TA-ERI seems to be able to suppress all atrial arrhythmias in a subset of patients suffering from both Afl and Afib.

914 Antiarrhythmic Drugs

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Anaheim Convention Center, Hall E
Presentation Hour: 5:00 p.m.–7:00 p.m.

914-129 Is Procainamide Failure at Electrophysiologic Testing Really an Appropriate Marker for Drug-Resistant Ventricular Tachyarrhythmias

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Almost 2 decades ago, failure of intravenous procainamide (PA) to suppress inducible ventricular tachyarrhythmias at electrophysiologic testing (EP) was proposed as a marker for antiarrhythmic drug resistance in general [essentially all were class I]; this concept still exists as evidenced by its use in the MADIT protocol. To determine whether it is still valid, however, in an era where antiarrhythmics other than class I exist, we analyzed serial drug responsiveness in the ESVEM database. Specifically, drug efficacy (E) following prior drug failure (F) was determined, where 1st or subsequent drug, given in random sequence, included: quinidine, PA, imipramine, pirmenol, mexiletine, propefenone, d, l-sotalol (Sot). Respective E rates (as %) as 1st/2nd drug were: 33/17, 35/21, 17/13, 29/19, 39/25, 24/25, 40/46. Prior drug F reduced E for all except propafenone and Sot. Efficacy rates by class in the absence/presence of prior drug F were: IA:33/26; IB:42/38; IC:34/20. Sot E after F of single class I/multiple class I/any class IA or III were: 47%/46%/45%. Efficacy of class I after prior Sot F was only 8.6%. These results suggest that: (1) class I E declines after prior drug F, especially after Sot, but class I F is not highly predictive of Sot F; (2) Sot would be a better screening drug than PA.

914-130 Acute Effect of Amiodarone and Desethylamiodarone on Defibrillation Threshold

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Chronic oral amiodarone (AM), but not acute intravenous AM has been previously reported to raise defibrillation threshold (DFT). We hypothesize that the increase in DFT following chronic AM administration is related to the effect of its active metabolite, desethylamiodarone (DEA). Therefore, we compared the effect of intravenous AM or DEA or vehicle (V) on DFT in 24 anesthetized farm pigs in a random and blinded manner. Defibrillation was delivered through a transvenous tripolar lead (CPI 0060) using biphasic shock waveform. DFT was determined using an up-down algorithm protocol and was defined as the average minimal energy (J) delivered from ascending and descending serial shocks resulting in successful defibrillation. DFT together with drug concentration (Conc) was obtained at baseline and following a 10 mg/kg dose or equivalent volume of V. *Results:* are shown below:

	AM (n = 8)	DEA (n = 8)	V (n = 8)
Baseline (J)	22.7 ± 4.1	20.5 ± 6.3	26.8 ± 7.7
Postdose (J)	26.1 ± 2.9	33.9 ± 13.6*	23.1 ± 7.4
Conc. (μg/ml)	0.72 ± 0.46	0.49 ± 0.29	0 ± 0

*p < 0.05 vs baseline

No DEA Conc. was detectable post intravenous AM administration.

Conclusion: Acute administration of intravenous desethylamiodarone significantly increased defibrillation threshold, whereas intravenous amiodarone showed a minimal change. These findings support the hypothesis that desethylamiodarone can contribute significantly to DFT elevation following chronic amiodarone administration.

914-131 Effects of Amiodarone on the Circadian Pattern of Sudden Cardiac Death. Results from the Congestive Heart Failure-Survival Trial of Antiarrhythmic Therapy

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Background: The circadian distribution of the onset of sudden cardiac death (SCD) has been shown to be influenced by beta-blockade (BHAT) and class I antiarrhythmic drugs (CAST). However, the effect of amiodarone on the circadian variation of SCD remains unknown. We retrospectively analyzed the circadian pattern of SCD in the Congestive Heart Failure-Survival Trial of Antiarrhythmic Therapy (CHF-STAT) which was a double-blind randomized